$%^Dialog;HighlightOn=*;HighlightOff=*;

Connecting via Winsock to Dialog

Logging in to Dialog

Trying 31060000009999...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
****
ENTER PASSWORD:
****
Welcome to DIALOG

Dialog level 02.12.60D

Last logoff: 06apr03 10:43:16
Logon file405 17apr03 13:29:33
*** ANNOUNCEMENT ***
***
? b 411
17apr03 13:29:52 User217743 Session D602.2
$0.00 0.073 DialUnits File410
$0.00 Estimated cost File410
$0.07 TELNET
$0.07 Estimated cost this search
$0.07 Estimated total session cost 0.238 DialUnits
File 411:DIALINDEX(R)

DIALINDEX(R)
(c) 2003 The Dialog Corporation plc

*** DIALINDEX search results display in an abbreviated
*** *** format unless you enter the SET DETAIL ON
command. *** ? set files biochem
>>> 68 does not exist
>>> 162 is unauthorized
>>>2 of the specified files are not available
You have 21 files in your file list.
(To see banners, use SHOW FILES command)
? s (gene()therapy) and (dog or canine or canid)

Your SELECT statement is:
s (gene()therapy) and (dog or canine or canid)

Items File
----- ----
372 5: Biosis Previews(R)_1969-2003/Apr W2
329 34: SciSearch(R) Cited Ref
Sci_1990-2003/Apr W2 32 50: CAB
Abstracts_1972-2003/Mar
23 65: Inside Conferences_1993-2003/Apr
W2 116 71: ELSEVIER
BIOBASE_1994-2003/Apr W2
240 73: EMBASE_1974-2003/Apr W2
28 94: JICST-EPlus_1985-2003/Apr W2
27 98: General Sci
Abs/Full-Text_1984-2003/Mar 3 103: Energy
SciTec_1974-2003/Apr B1
3 143: Biol. & Agric. Index_1983-2003/Mar
129 144: Pascal_1973-2003/Apr W1
197 155: MEDLINE(R)_1966-2003/Apr W2
30 156: ToxFile_1965-2003/Apr W1
8 172: EMBASE Alert_2003/Apr W2
3 369: New Scientist_1994-2003/Apr W1
2 370: Science_1996-1999/Jul W3
188 399: CA SEARCH(R)_1967-2003/UD=13816
2 434: SciSearch(R) Cited Ref
Sci_1974-1989/Dec
18 files have one or more items; file list includes 21
files.
? rf
Your last SELECT statement was:
S (GENE()THERAPY) AND (DOG OR CANINE OR
CANID)

Ref Items File
--- ----- ----
N1 372 5: Biosis Previews(R)_1969-2003/Apr
W2 N2 329 34: SciSearch(R) Cited Ref
Sci_1990-2003/Apr W2 N3 240 73:
EMBASE_1974-2003/Apr W2
N4 197 155: MEDLINE(R)_1966-2003/Apr W2
N5 188 399: CA
SEARCH(R)_1967-2003/UD=13816
N6 129 144: Pascal_1973-2003/Apr W1
N7 116 71: ELSEVIER BIOBASE_1994-2003/Apr
W2
N8 32 50: CAB Abstracts_1972-2003/Mar
N9 30 156: ToxFile_1965-2003/Apr W1
N10 28 94: JICST-EPlus_1985-2003/Apr W2
18 files have one or more items; file list includes 21
files.
- Enter P or PAGE for more -
? b 155
17apr03 13:30:59 User217743 Session D602.3
$3.14 1.571 DialUnits File411
$3.14 Estimated cost File411
$0.46 TELNET
$3.60 Estimated cost this search
$3.67 Estimated total session cost 1.809 DialUnits
File 155:MEDLINE(R) 1966-2003/Apr W2
(c) format only 2003 The Dialog Corp.
*File 155: Medline has been reloaded and accession
numbers have changed. Please see HELP NEWS 155.

? s (gene()therapy) and (dog or canine or canid)/ti
609714 GENE
1919497 THERAPY
20528 GENE(W)THERAPY
28122 DOG/TI
23906 CANINE/TI
22 CANID/TI
S1 82 (GENE()THERAPY) AND (DOG OR CANINE

OR CANID)/TI ? s (gene()therapy)/ab and (dog or canine or canid)/ti
439457 GENE/AB
423082 THERAPY/AB
11131 GENE/AB(W)THERAPY/AB
28122 DOG/TI
23906 CANINE/TI
22 CANID/TI
S2 66 (GENE()THERAPY)/AB AND (DOG OR CANINE OR CANID)/TI
? t s2/free/all

? t s2/3,ab/2,8,13,32,55

2/3,AB/2
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

14648496 22471772 PMID: 12406898
Therapeutic factor VIII levels and negligible toxicity in mouse and *dog* models of hemophilia A following gene therapy with high-capacity adenoviral vectors.
Chuah Marinee K L; Schiedner Gudrun; Thorrez Lieven; Brown Brian; Johnston Marion; Gillijns Veerle; Hertel Sabine; Van Rooijen Nico; Lillicrap David; Collen Desire; VandenDriessche Thierry; Kochanek Stefan Center for Transgene Technology and Gene Therapy, Flanders Interuniversity Institute for Biotechnology, University of Leuven, Belgium. Blood (United States) 10 24 2002, 101 (5) p1734-43, ISSN 0006-4971 Journal Code: 7603509
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
High-capacity adenoviral (HC-Ad) vectors expressing B-domain-deleted human or canine factor VIII from different liver-specific promoters were evaluated for *gene* *therapy* of hemophilia A. Intravenous administration of these vectors into hemophilic FVIII-deficient immunodeficient SCID mice (FVIIIKO-SCID) at a dose of 5 x 10(9) infectious units (IU) resulted in efficient hepatic gene delivery and long-term expression of supraphysiologic FVIII levels (exceeding 15 000 mU/mL), correcting the bleeding diathesis. Injection of only 5 x 10(7) IU still resulted in therapeutic FVIII levels. In immunocompetent hemophilic FVIII-deficient mice (FVIIIKO), FVIII expression levels peaked at 75 000 mU/mL but declined thereafter because of neutralizing anti-FVIII antibodies and a cellular immune response. Vector administration did not result in thrombocytopenia, anemia, or elevation of the proinflammatory cytokine interleukin-6 (IL-6) and caused no or only transient elevations in serum transaminases. Following transient in vivo depletion of macrophages before gene transfer, significantly higher and stable FVIII expression levels were observed. Injection of only 5 x 10(6) HC-Ad vectors after macrophage depletion resulted in long-term therapeutic FVIII levels in the FVIIIKO and FVIIIKO-SCID mice. Intravenous injection of an HC-Ad vector into a hemophilia A dog at a dose of 4.3 x 10(9) IU/kg led to transient therapeutic canine FVIII levels that partially corrected whole-blood clotting time. Inhibitory antibodies to canine FVIII could not be detected, and there were no signs of hepatotoxicity or of hematologic abnormalities. These results contribute to a better understanding of the safety and efficacy of HC-Ad vectors and suggest that the therapeutic window of HC-Ad vectors could be improved by minimizing the interaction between HC-Ad vectors and the innate immune system.

2/3,AB/8
DIALOG(R)File 155:MEDLINE(R)
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14287753 22294654 PMID: 12407379
Gene transfection of hepatocyte growth factor attenuates cardiac remodeling in the *canine* heart: A novel gene therapy for cardiomyopathy.
Ahmet Ismayil; Sawa Yoshiki; Iwata Keiji; Matsuda Hikaru; et al First Department of Surgery, Osaka University Medical School, Osaka, Japan.
Journal of thoracic and cardiovascular surgery (United States) Nov 2002, 124 (5) p957-63, ISSN 0022-5223 Journal Code: 0376343 Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
OBJECTIVE: Hepatocyte growth factor, a potent angiogenic agent, is unique in having the effects of antiapoptosis and antifibrosis. In the present study we used the rapid pacing-induced heart failure canine model to investigate the effect of gene transfection of hepatocyte growth factor on the failing heart. METHODS: Four weeks after onset of rapid pacing, either the human hepatocyte growth factor gene (160 microg; hepatocyte growth factor group, n = 7) or empty vector (control group, n = 7) was directly injected into the left ventricular myocardium by means of the hemagglutinating virus of Japan liposome method. RESULTS: At 4 weeks after gene transfection, the left ventricular global function, assessed by means of pressure-volume loop analysis, was improved in the hepatocyte growth factor group as preload-recruitable stroke work (percentage of baseline: 80% +/- 20% from 38% +/- 15% before gene transfection, P =.005), whereas it was not changed in the control group (50% +/- 18% from 50% +/- 18%). Weekly echocardiography showed that this improvement began in the week after gene transfer. The hearts in the hepatocyte growth factor

group had a large wall thickness, large myocyte diameter, high capillary density, low fibrotic area fraction, and low density of apoptotic nuclei revealed by means of histologic analysis compared with that in the control group. Myocardial perfusion flow, assessed with color microspheres, was increased in the hepatocyte growth factor group (percentage of baseline: 79% +/- 16% from 48% +/- 14%, P =.010), whereas it was reduced in the control group (30% +/- 12% from 45% +/- 17%). CONCLUSIONS: Gene transfection of hepatocyte growth factor promoted angiogenesis, improved perfusion, decreased fibrosis and apoptosis, promoted recovery from myocyte atrophy, and thereby attenuated cardiac remodeling and improved myocardial function in the failing heart. It is a novel *gene* *therapy* for human heart failure.

2/3,AB/13
DIALOG(R)File 155:MEDLINE(R)
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11663371 99098310 PMID: 9883841
Correction of hemophilia B in *canine* and murine models using recombinant adeno-associated viral vectors.
Snyder R O; Miao C; Meuse L; Tubb J; Donahue B A; Lin H F; Stafford D W; Patel S; Thompson A R; Nichols T; Read M S; Bellinger D A; Brinkhous K M; Kay M A
Cell Genesys Inc., Foster City, California 94404, USA.
Nature medicine (UNITED STATES) Jan 1999, 5 (1) p64-70, ISSN 1078-8956 Journal Code: 9502015
Contract/Grant No.: HL01648; HL; NHLBI; HL53682; HL; NHLBI Comment in Nat Med. 1999 Jan;5(1) 21-2; Comment in PMID 9883831 Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Hemophilia B, or factor IX deficiency, is an X-linked recessive disorder occurring in about 1 in 25,000 males. Affected individuals are at risk for spontaneous bleeding into many organs; treatment mainly consists of the transfusion of clotting factor concentrates prepared from human blood or recombinant sources after bleeding has started. Small- and large-animal models have been developed and/or characterized that closely mimic the human disease state. As a preclinical model for *gene* *therapy*, recombinant adeno-associated viral vectors containing the human or canine factor IX cDNAs were infused into the livers of murine and canine models of hemophilia B, respectively. There was no associated toxicity with infusion in either animal model. Constitutive expression of factor IX was observed, which resulted in the correction of the bleeding disorder over a period of over 17 months in mice. Mice with a steady-state concentration of 25% of the normal human level of factor IX had normal coagulation. In hemophilic dogs, a dose of rAAV that was approximately 1/10 per body weight that given to mice resulted in 1% of normal canine factor IX levels, the absence of inhibitors, and a sustained partial correction of the coagulation defect for at least 8 months.

2/3,AB/32
DIALOG(R)File 155:MEDLINE(R)
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10849506 97200823 PMID: 9048652
Expression and function of recombinant endothelial nitric oxide synthase gene in *canine* basilar artery.
Chen A F; O'Brien T; Tsutsui M; Kinoshita H; Pompili V J; Crotty T B; Spector D J; Katusic Z S
Department of Anesthesiology, Mayo Clinic, Rochester, Minn 55905, USA. Circulation research (UNITED STATES) Mar 1997, 80 (3) p327-35, ISSN 0009-7330 Journal Code: 0047103
Contract/Grant No.: GM-08288; GM; NIGMS; HL-44116; HL; NHLBI; HL-53542; HL; NHLBI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Endothelial NO synthase (eNOS) is an enzyme responsible for the production of a potent vasodilator and a key regulator of vascular tone, NO. In peripheral arteries, expression of a recombinant eNOS gene increases production of NO in the blood vessel wall. This approach appears to be a promising strategy for *gene* *therapy* of cerebrovascular disease. The major objective of the present study was to determine whether a recombinant eNOS gene (AdCMVNOS) can be functionally expressed in cerebral arteries. Replication-defective recombinant adenovirus vectors encoding bovine eNOS and Escherichia coli beta-galactosidase (AdCMVLacZ) genes, driven by the cytomegalovirus promoter, were used for ex vivo gene transfer. Rings of canine basilar artery were incubated with increasing titers of the vectors in MEM. Twenty-four or forty-eight hours after gene transfer, expression and function of AdCMVNOS were evaluated by (1) immunohistochemical staining, (2) isometric tension recording, and (3) cGMP radioimmunoassay. Transfection with AdCMVNOS resulted in the expression of recombinant eNOS protein in the vascular adventitia and endothelium, associated with significantly reduced contractile responses to UTP and enhanced endothelium-dependent relaxation to calcium ionophore A23187. Basal production of cGMP was significantly increased in the transfected vessels. The reduced contractions to UTP with increased cGMP production were reversed by a NOS inhibitor, N(G)-monomethyl-L-arginine. Contractions to UTP or production of cGMP were not affected in arteries transfected with AdCMVLacZ reporter gene. The results of the present study represent the first successful transfer and functional expression of

recombinant eNOS gene in cerebral arteries. Our findings suggest that cerebral arterial tone can be modulated by recombinant eNOS expression in the vessel wall.

2/3,AB/55
DIALOG(R)File 155:MEDLINE(R)

08474122 95162338 PMID: 7858769
Lipofectin-mediated versus adenovirus-mediated gene transfer in vitro and in vivo: comparison of *canine* and porcine model systems. Mazur W; Ali N M; Grinstead W C; Schulz D G; Raizner A E; French B A Department of Medicine, Methodist Hospital, Houston, Texas. Coronary artery disease (UNITED STATES) Sep 1994, 5 (9) p779-86, ISSN 0954-6928 Journal Code: 9011445
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
BACKGROUND: Restenosis after coronary angioplasty might be prevented by locally delivered *gene* *therapy* in conjunction with percutaneous transluminal coronary angioplasty (PTCA), since this approach should provide a sustained source of therapeutic protein within the dilated lesion. However, the potential application of *gene* *therapy* is limited by the technical barrier of efficiently transferring genes to vascular cells. METHODS: We used cultured coronary smooth muscle cells of human, porcine, and canine origin to evaluate three methods of gene transfer: recombinant adenovirus, liposomal complexes (Lipofectin), and Lipofectin supplemented with hemagglutinin. We then compared Lipofectin- and adenovirus-mediated direct gene transfer in canine and porcine coronary arteries. RESULTS: The lipofection of cultured smooth muscle cells was enhanced by adding hemagglutinin, yielding luciferase levels that were 631-fold (human), ninefold (porcine), and sevenfold (canine) higher than with Lipofectin alone. However, the recombinant adenovirus directed even higher levels of gene expression, yielding luciferase levels that were 113,000-fold (human), 450-fold (porcine), and 230-fold (canine) higher than with Lipofectin alone. After percutaneous transluminal local delivery to intact canine coronary arteries, the adenovirus produced 55 times more luciferase than did Lipofectin. In living porcine coronary arteries, adenovirus produced 95 times more luciferase than did Lipofectin. CONCLUSION: Recombinant adenovirus produces far more recombinant protein than does Lipofectin after percutaneous transluminal direct gene transfer to canine and porcine coronary arteries. Adenoviral vectors may therefore prove useful in evaluating the potential of *gene* *therapy* in large animal models of coronary restenosis.
? logoff
17apr03 13:36:14 User217743 Session D602.4
$10.71 3.348 DialUnits File155
$0.00 66 Type(s) in Format 8
$1.05 5 Type(s) in Format 4 (UDF)
$1.05 71 Types
$11.76 Estimated cost File155
$1.40 TELNET
$13.16 Estimated cost this search
$16.83 Estimated total session cost 5.156 DialUnits
Logoff: level 02.12.60 D 13:36:14